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Terbinafine-induced lichenoid drug eruption: case report and review of terbinafine-associated cutaneous adverse events

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Abstract

Terbinafine is an antifungal agent used in the treatment of hair, nail, and skin dermatophyte infections. Skin side effects to terbinafine are not common. Lichenoid drug eruption is a medication-related adverse cutaneous event; the lesion morphology and pathology mimic lichen planus. A woman with onychomycosis developed a lichenoid drug eruption one week after starting terbinafine. The features of her dermatosis and the characteristics of two additional men who also experienced terbinafine-induced lichenoid drug eruption are discussed. They were receiving a daily terbinafine dosage of either 125mg or 250mg to treat onychomycosis or tinea cruris. The lichenoid drug eruption presented as diffuse or symmetric lesions within one to two weeks after starting terbinafine treatment. The extremities, chest, abdomen, and/or trunk were common sites. Less frequent locations were the lips, nails, palms, soles, and suprapubic region; lesions did not occur on the oral or genital mucosa. The eruption resolved after discontinuation of the medication (with or without treatment using topical corticosteroids, systemic corticosteroids, or both). In addition, more frequently occurring terbinafine-associated cutaneous adverse events (such as urticaria, erythematous eruptions, pruritus, acute generalized exanthematous pustulosis, subacute cutaneous lupus erythematosus, and papulosquamous conditions) are reviewed.

Keywords: adverse cutaneous, drug, effect, event, fungal, lichenoid, reaction, side, skin, terbinafine

Introduction

Terbinafine, an allylamine, is an antifungal agent used for the management of onychomycosis [1-13]. A lichenoid drug eruption, also referred to as a lichen planus-like eruption, can present with similar features that are observed in patients with idiopathic lichen planus [14-18]. A woman who developed a lichenoid drug reaction to terbinafine is described and terbinafine-associated adverse cutaneous events are reviewed.

Case Synopsis

A 68-year-old woman presented to her primary care physician with dystrophy of her toenails. Therapy was initiated with oral terbinafine at a dosage of 250mg daily. She began to develop an itchy rash on her lower abdomen and groin areas after the seventh day of treatment; however, she continued to take the medication for an additional seven days. The rash continued to spread to her chest after she discontinued the terbinafine. One week after stopping the terbinafine she presented for evaluation of her skin. Cutaneous examination showed pruritic erythematous scaly plaques on her chest, abdomen, and suprapubic region (**Figure 1**). Skin biopsies of lesions on her right chest and right abdomen were performed.

Microscopic examination of both biopsies had similar pathologic changes (**Figure 2**). The epidermis shows compact hyperkeratosis, acanthosis and hypergranulosis; there are also focal areas of

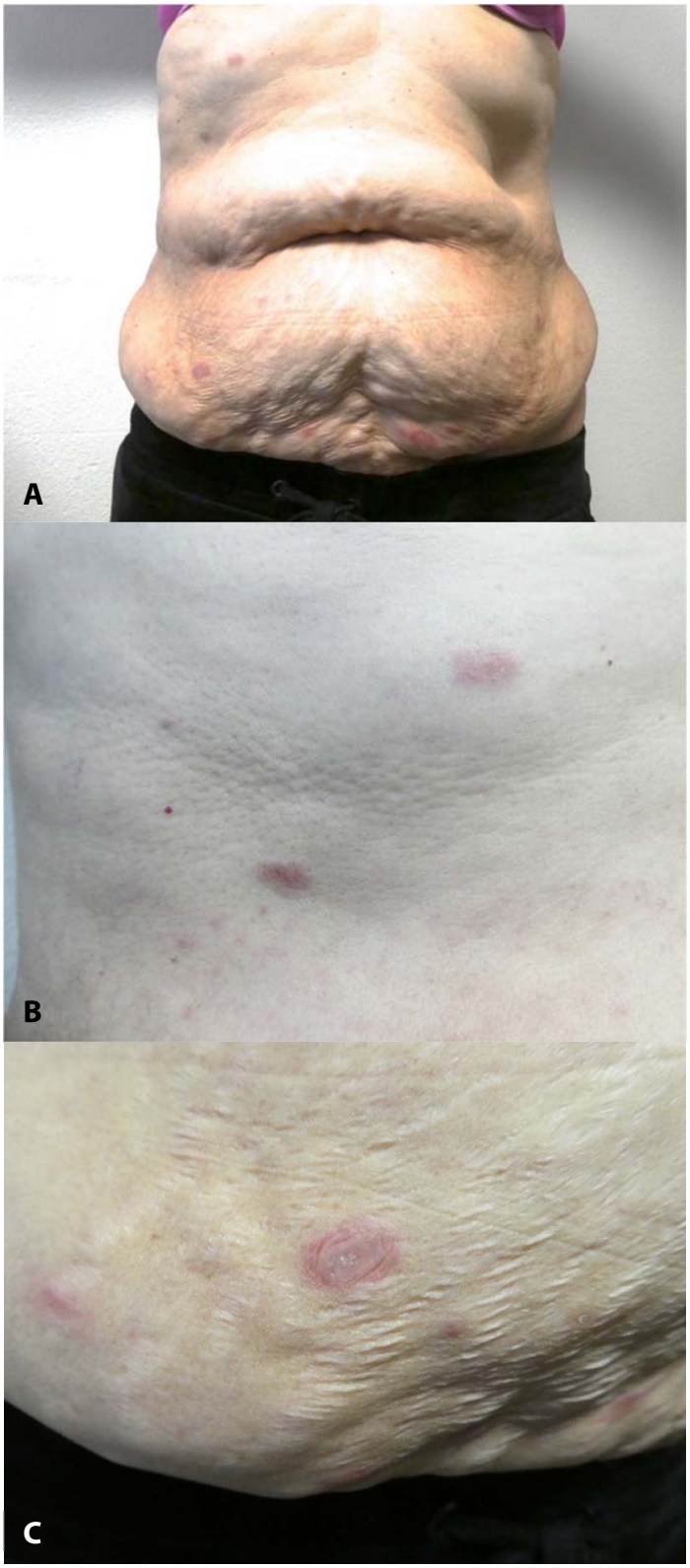

Figure 1. *A-C). Distant (A) and closer (B, **C)** views of terbinafine-induced lichenoid drug eruption. Symmetrically distributed pruritic erythematous scaly plaques on the chest (A, **B)** and abdomen (A, **C)** of a 68-year-old woman that appeared one week after initiating terbinafine therapy (at a daily dosage of 250mg) to treat onychomycosis.*

spongiosis, scattered necrotic keratinocytes, and vacuolar alteration of the basal layer. In the upper dermis, there is not only an interface dermatitis with a band-like infiltration of lymphocytes and histiocytes, but also perivascular inflammation and extravasated erythrocytes; in addition, there are numerous eosinophils. The pathologic changes are those of a lichenoid dermatitis. The presence of abundant eosinophils is consistent with the possibility of a drug-related etiology. Correlation of the clinical history, the lesion morphology, and the pathologic changes establish the diagnosis of terbinafine-induced lichenoid drug eruption.

Terbinafine had already been discontinued a week earlier. However, the patient was still developing new symptomatic lesions. Management included systemic prednisone (60mg daily for six days,

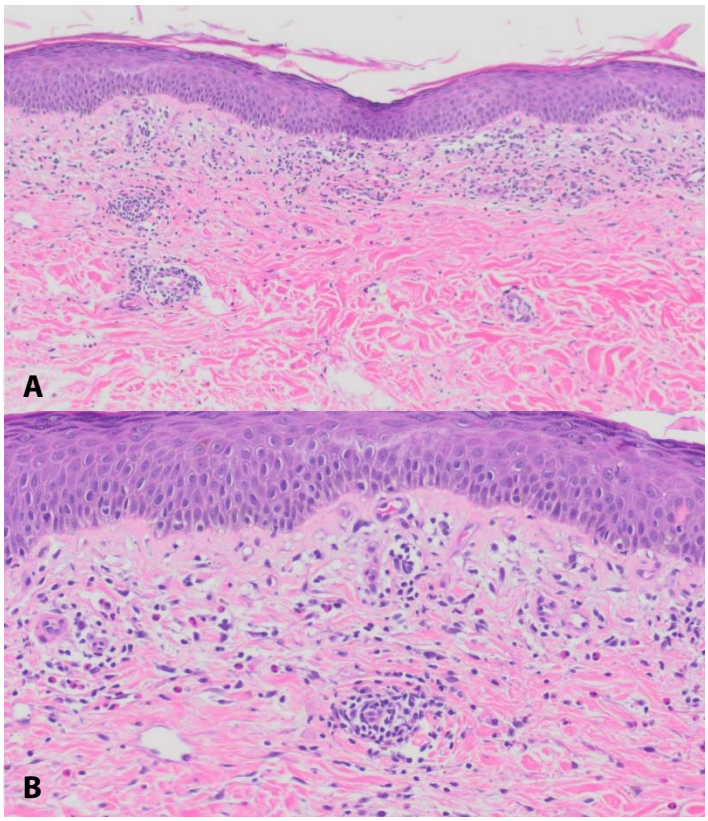

Figure 2. ***A, B)***. *Distant (**A**) and closer (**B**) views of microscopic features of terbinafine-induced lichenoid drug eruption. There is compact hyperkeratosis and hypergranulosis. There are focal areas of spongiosis in the lower layers of the acanthotic epidermis. Vacuolar alteration of the epidermal basal layer is also present. A band-like infiltrate of lymphocytes, histiocytes and numerous eosinophils fill the papillary dermis; in addition, there is perivascular inflammation in the upper dermis. H&E, **A)** 10×; **B)** 20×.*

followed by 40mg daily for four days, followed by 20mg daily for two days) and topical betamethasone dipropionate 0.05% cream three times daily to the lesions. The patient returned two weeks later; the pruritus had resolved and her skin lesions had completely cleared. She decided not to treat her onychomycosis. There was no recurrence of the dermatosis at follow-up examination two months later.

Case Discussion

Terbinafine interferes with fungal cell membrane ergosterol biosynthesis by selectively inhibiting the fungal enzyme squalene epoxidase [1-13]. Systemic dosing of the drug is a mainstay treatment of fungal nail infection and dermatophytic hair and skin infections [1-13]. Gastrointestinal symptoms (nausea, diarrhea, dyspepsia and abdominal pain) and headaches are the main adverse events associated with terbinafine [1-13]. However, a unique terbinafine-related side effect is taste disturbance [19-22].

Adverse cutaneous events to terbinafine have been observed in approximately two percent of patients (**Box 1**), [23-67]. The most common skin side effects include urticaria, erythematous dermatoses, and pruritus [13, 29, 36, 50]. However, albeit uncommon, life threatening conditions such as Stevens-Johnson syndrome and toxic epidermal necrolysis have been reported [35, 57, 66].

Acute generalized exanthematous pustulosis is an uncommon cutaneous drug reaction characterized by the acute onset of widespread small nonfollicular intraepidermal or subcorneal sterile pustules arising on edematous and erythematous skin. The patients typically also have pruritus, fever, and neutrophilia. This dermatosis has been associated with several antibiotics such as ampicillin, amoxicillin, macrolides, quinolones, and sulfonamides. Acute generalized exanthematous pustulosis usually resolves either spontaneously or with corticosteroid therapy within two weeks of discontinuing the causative drug [23].

Acute generalized exanthematous pustulosis has been described in at least 27 terbinafine-treated patients; the male to female ratio is 1:1 [23-28]. The

Box 1. *Cutaneous adverse events associated with terbinafine.*

Acute generalized exanthematous pustulosis [23-28]
Alopecia areata [29]
Dermatitis-exacerbation [30]
Dermatomyositis [31, 32]
Desquamation [33]
Erythema annulare-like psoriatic drug eruption [34]
Erythema multiforme [35-39]
Erythema nodosum [40]
Erythematous eruption [29]
Erythroderma [36][a]
Fixed drug eruption [41]
Gray facial hyperpigmentation [42][b]
Hairloss [29]
Hypersensitivity syndrome [43]
Lichenoid drug eruption [44, 45, current report]
Pityriasis rosea-like eruption [36, 46]
Pruritus [29]
Psoriasis—de novo or exacerbation [26, 28, 36, 47-49]
Rash [50]
Rowell syndrome [51-54][c]
Skin eruption-mild [55]
Skin rash-severe [56]
Stevens-Johnson syndrome [57]
Subacute cutaneous lupus erythematosus [58-61]
Symmetric drug-related intertriginous and flexural exanthema [62-64][d]
Systemic lupus erythematosus [57, 65]
Toxic epidermal necrolysis [35, 66]
Urticaria [36, 50, 67][e]

[a] *A 56-year-old woman developed generalized pruritic erythroderma on day 27 after starting terbinafine; she was treated with oral prednisone and severe desquamation occurred. The erythroderma and desquamation resolved over two weeks; however, she developed biopsy-proven warts and seborrheic keratoses all over her body.*

[b] *A 65-year-old man developed gray-brown macula on his face beginning four weeks after starting terbinafine for onlychomycosis. Melanin localized within macrophages in both the upper and lower dermis was found on microscopic examination of the biopsy.*

[c] *Rowell syndrome, described in 1963, is characterized by lupus erythematosus with erythema multiforme-like lesions and specific immunological abnormalities: a positive rheumatoid factor, a speckled antinuclear antibody and precipitating antibodies to saline extract of human tissue (anti-SjT). Subsequently, in 2000, major (clinical features of a lupus erythematosus subtype, erythema multiforme-like lesions, and a speckled pattern of antinuclear antibody) and minor [chilblains, anti-Sjogren syndrome antigen A (SS-A; also referred to as Ro) and anti-Sjogren syndrome antigen B (SS-B; also referred to as La) antibodies, and a positive rheumatoid factor]; all three major criteria and at least one of the minor criteria are necessary to establish the diagnosis of RS.*

[d] *Symmetric drug-related intertriginous and flexural exanthema (SDRIFE) is also referred to as the non-contact allergic drug-induced variant of baboon syndrome; the latter is a descriptive term that has been used when a patient develops erythematous buttocks similar in appearance to those of a baboon. SDRIFE has most commonly been associated with antibiotics such as penicillins and beta-lactams.*

[e] *This includes a 25-year-old woman in whom phototesting confirmed ultraviolet B-sensitive urticarial reaction that occurred within minutes after sun exposure while she was taking terbinafine for onychomycosis.*

onset age ranged from 20 to 74 years (median, 57 years). The latency period between starting terbinafine and the onset of acute generalized exanthematous pustulosis ranged from one day to 44 days (median 9 days), [23].

Terbinafine can also be associated with the new onset or flaring of connective tissue disorders. The drug can cause or flare dermatomyositis [31, 32]. In addition, terbinafine is one of the more frequent culprits of drug-induced subacute cutaneous lupus erythematosus [58-61]. It has also been associated with the exacerbation of systemic lupus erythematosus [57, 65] and Rowell syndrome [51-54].

Several papulosquamous conditions can also be induced by terbinafine. Psoriasis can be exacerbated or initially present following treatment with terbinafine [26, 28, 36, 47-49]. In addition, terbinafine can flare dermatitis [30] or result in a pityriasis rosea-like eruption [36, 46].

Lichenoid drug eruption morphologically and pathologically mimics lichen planus. In contrast to lichen planus, lichenoid drug eruption is more likely to be symmetric with photodistributed lesions that are larger and more psoriasiform without Wickham striae or oral mucosa involvement. In addition, eosinophils may be an accompanying histologic feature. Several medications have been associated with lichenoid drug eruption. However, this cutaneous adverse event is an uncommon occurrence in patients receiving terbinafine [14-18].

Including the reported patient, to the best of our understanding, only three individuals with a lichenoid eruption related to terbinafine have been described (**Table 1**), [44, 45]. Two are men and one is a woman. They range in age from 22 years to 73 years (median, 68 years).

The conditions being treated with terbinafine were either tinea ungium (2 patients) or tinea cruris (1 man). The 73-year-old man with onychomycosis was being treated with 125mg of terbinafine daily. In contrast, the other two patients were receiving 250mg of terbinafine daily. Both patients being treated with the higher dosage of terbinafine experienced pruritus. The other individual did not have any itching. The onset of lichenoid drug eruption after starting terbinafine ranged from one week (the 68-year-old woman) to two weeks (both men). The skin lesions were symmetrically distributed in the woman and the older man. In contrast, they were diffusely located on the 22-year-old man and appeared as lichenoid papules initially on his trunk. Subsequently, they spread to involve his arms and legs, palms and soles, lip, and thumb nails.

The skin lesions appeared as violaceous and/or erythematous scaly plaques on the two older patients. They presented on the abdomen and suprapubic region in the woman and spread to also affect her chest. The plaques started on the legs of the older man and then spread to his trunk and arms. For all patients with lichenoid drug eruption, none had lesions that affected the oral or genital mucosa. Also, none of the lesion demonstrated Wickham striae. All patients had biopsies of their skin lesions to confirm the diagnosis of terbinafine-induced lichenoid drug eruption.

The initial management for patients with terbinafine-induced lichenoid drug eruption was discontinuation of the medication. The skin lesions promptly resolved after stopping the terbinafine in the older man. Surprisingly, his lichenoid drug eruption did not recur on repeat challenge with the drug. The younger patients were treated with corticosteroid after stopping the terbinafine. The woman received oral prednisone and topical betamethasone dipropionate. The younger man received systemic prednisolone, oral antihistamines, and topical tacrolimus 0.1% cream. The reported woman's pruritus and skin lesions both resolved within two weeks and did not recur. The younger man's symptoms and skin lesions completely resolved within 8 weeks. His nail changes cleared after 12 weeks of treatment.

Conclusion

Cutaneous adverse events related to terbinafine are not common; however, they do occasionally occur. In addition to urticaria, erythematous eruptions, and pruritus, terbinafine-induced acute generalized exanthematous pustulosis, subacute cutaneous lupus erythematosus, and papulosquamous

Table 1. *Characteristics of patients with terbinafine-induced lichenoid drug eruption.*

C	A G	Tbf Dosa	Cond	On	Pr	Morph	Loc	MI	Bx	Tx	Response	Ref
1[a]	22 M	250	TCC	2	+	Diffuse, Lichenoid papules	Ext, Lip, P&S, TN, Tr	–	+	SD, Plne, Tac, Ah	8 wk & 12 wk[b]	44
2[c]	68 W	250	TUC	1	+	Symmetric, Erythmtous scaly plaques	Abd, Ch, Sp	–	+	SD, Pred, Beta	2 wk[d]	CR
3[e]	73 M	125	TUN	2	–	Symmetric, Erythmtous violaceous scaly plaques	Arm, Leg, Tr	–	+	SD	PR[f]	45

Abbreviations: A, age (years); Abd, abdomen; Ah, antihistamine; Beta, betamethasone dipropionate 0.05% cream thrice daily for two wk; Bx, biopsy confirmation—hematoxylin and eosin stained sections--of LDE; C, case; Ch, chest; Cond, condition being treated with tbf; CR, current report; Dosa, dosage (mg per day); Erythmtous, erythematous; Ext, extremities; G, gender; KOH, potassium hydroxide; LDE, lichenoid drug eruption; Loc, location of skin lesions; M, man;mg, milligrams; MI, mucosal (roal and genital) involvement; Morph, morphology of LDE skin lesions; LTT, lymphocyte transformation test; On, onset of lichenoid drug eruption after starting terbinafine (weeks); Plne, prednisolone (30mg per day, duration not stated); Pr, pruritus; PR, prompt resolution of LDE after stopping tbf; Pred, prednisone (60mg for 6 days, then 40mg for four days, and then 20mg for two days); P&S, palms and soles; Ref, reference; Response, response to tx; SD, stop drug; Sp, suprapubic; Tac, tacrolimus 0.1% cream once daily; duration not stated); Tbf, terbinafine; TCC, tinea cruris confirmed by KOH-positive skin fungal microscopic test; TN, thumb nail; Tr, trunk; TUC, tinea unguim confirmed by periodic acid-Schiff stain-positive right great toenail clipping and Trichophyton rubrum positive nail plate culture; TUN, tinea unguim not confirmed by objective testing; Tx, treatment; W, woman; wk, weeks; +, present; –, absent; &, and.

[a]The patient has a 15 year history of untreated vitiligo. He presented with KOH-positive tinea cruris and started on terbinafine. An itchy papular eruption appeared on his trunk two weeks after starting terbinafine; during the next three weeks, the skin lesions spread to his exrtremities, palms and soles, lips and thumb nails. He presented three weeks later (still on daily terbinafine) for evaluation.

[b]After 8 weeks of treatment, the lesion on his body and lips completely cleared; after 12 weeks of treatment, the nail changes disappeared.

[c]The rash began on her abdomen one week after starting terbinafine. She continued to take the medication daily for another 7 days and the rash spread to her chest and suprapubic area. She was seen for evaluation one week after stopping terbinafine; the rash persisted and new lesions were appearing.

[d]Her pruritus and dermatosis both resolved prior to her two week follow-up visit; there was no recurrence at two months follow-up.

[e]The rash appeared on his legs 5 weeks after starting ticlopidine hydrochloride (200mg per day) and two weeks after starting terbinafine (125mg per day). He was seen three months after the onset of the rash; the skin lesions had spread to his trunk and arms. He stopped the ticlopidine hydrochloride and the rash continued for the next two weeks; he then stopped the terbinafine and the skin lesions promptly resolved. The LTT was positive for terbinafine and negative for ticlopidine hydrochloride.

[f]One month after stopping terbinafine, an oral challenge test to the patient was performed with terbinafine 125mg daily for 7 days; there was no significant cutaneous adverse event and the LTT to terbinafine was negative. He was also challenged to ticlopidine hydrochloride; no skin side effects occurred and the LTT was also negative. The investigators speculated that, in this patient, tolerance to terbinafine might have been induced.

conditions have been observed. Terbinafine-associated lichenoid drug eruption is a rare skin side effect that has only been observed in three individuals: two men and one woman. They were being treated with terbinafine at a daily dosage of either 125mg (1 man) or 250mg (2 patients) for tinea cruris (1 man) or onychomycosis (2 patients). The lichenoid drug eruption appeared as diffuse (1 man) or symmetric (2 patients) lesions most commonly on the extremities, chest, abdomen, and/or trunk within one to two weeks after starting treatment with terbinafine. The lips, nails, palms, soles, and suprapubic region were less frequent sites; no lesions occurred on the oral or genital mucosa. The eruption resolved after discontinuation of the medication (with or without treatment using topical corticosteroids, systemic corticosteroids, or both).

Potential conflicts of interest

Dr. Cohen is a paid consultant for ParaPRO; however, this activity has no influence as a potential conflict of interest with regards to the manuscript. The remaining authors have no interests to report.

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